As regards changes which could lead to irreversible lowering of cardiac function, the arteritis might prove in the long run the most significant if it progressed. There is no evidence in the affected vessels of obstructive phenomena at the time of the patient's death and no effects on the myocardium were noted. It would seem reasonable to infer that if this necrotizing arteritis was a manifestation of graft rejection it would probably have progressed, as the patient had almost maximal treatment with the immunosuppressive drugs and procedures available today. But we cannot be certain at this stage that the arteritis was the result of rejection. It may be significant that the second heart transplant patient in Cape Town was alive and improving 126 days after transplantation at the time of writing, having had similar treatment throughout, apart from intensified precautions against infection. It would seem reasonable to presume that whatever rejection phenomena he may have developed during this period, an arteritis of the type and extent seen in the first human transplant has not so far developed.

Summary

Necropsy on the first heart transplantation in man confirmed that death was due to extensive bilateral pneumonia and not to rejection of the heart. Signs of rejection, however, were demonstrated histologically, but were of a low order compared with those seen in the transplanted dog heart. How far the mildness of the rejection signs is to be ascribed to species differences, immunosuppressive drugs and procedures, postoperative rest, and nursing and medical care, or to the good matching of leucocyte antigens cannot at the moment be assessed.

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Long-Term Hazards of Thorotrast

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For some 20 years after its introduction into clinical practice in 1928 a colloidal suspension of 232-thorium dioxide (Thorotrast) was extensively used as a radiographic contrast medium either to outline body cavities such as the renal pelvis or the cerebral ventricles or for the visualization of blood vessels. For all practical purposes thorium, once injected, is retained indefinitely by the body either locally at the site of injection, or, when given into the blood stream, mainly in reticuloendothelial cells in the liver, spleen, bone marrow, and lymph nodes. The effects of this retention are complex, and both chemical and radiation damage have to be considered, the former producing a slowly progressive tissue fibrosis, while the latter is reflected in an increased mortality from certain tumours. In considering the tumour hazard after the intra-arterial injection of Thorotrast account must be taken of the biological effects which may derive not only from 232-thorium deposited in soft tissues but also from its daughters, some of which become deposited in the organic and mineral components of bone and one of which, thoron, is excreted through the lungs. The late effects of increased body burdens of thorium include the development of tumours of the liver and spleen, leukaemia, aplastic anaemia, thrombocytopenia, and possibly carcinoma of the bronchus (da Silva Horta *et al.*, 1965).

Material and Methods

A search of departmental records allowed identification of patients receiving Thorotrast in the course of investigation in the Department of Surgical Neurology, Edinburgh.¹ All

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¹ Initial extraction of relevant case records was carried out by the late Mr. P. B. Ascroft as part of a preliminary study of the present series (Ascroft and Maccabe, 1962).

records relating to the years 1926 to 1955 inclusive were examined and yielded a total of 168 patients who had received Thorotrast either by intra-arterial injection (141 patients) or by direct injection into the cerebral ventricles or subarachnoid space (27 patients). Subsequent experience of these patients has been ascertained by follow-up inquiries directed towards tracing and establishing the current status of each individual. Wherever possible, surviving patients have been seen or, failing this, questionnaires have been sent to their medical practitioners. For patients found to be dead copies of death certificates have been obtained and case records relating to any deaths in hospital have been examined.

Results of Intra-arterial Injection of Thorotrast

Nine of the 141 patients in this group came from abroad for treatment and one further patient has since emigrated. These 10 patients have been excluded and are noted as such in Table I. All the remaining 131 patients were traced and their status was determined on 1 January 1966 or later. Twenty-two patients died within one year of Thorotrast administration as a direct result of the disease bringing them

TABLE I.—Distribution of 141 Patients Receiving Thorotrast Intra-arterially by Year of Injection, Age at Injection, and Volume Injected

Year of Injection	No. of Patients		Age at Injection (Years)	No. of Patients		Thorotrast Injected (ml.)	No. of Patients	
	Study Group	Excluded*		Study Group	Excluded*		Study Group	Excluded*
1930-4	6	1	0-4	1	—	0-9	—	—
1935-9	20	13	5-14	7	2	10-19	35	16
1940-4	52	7	15-24	25	3	20-29	33	6
1945-9	30	11	25-34	27	10	30-39	20	5
1950-4	—	—	35-44	28	8	40-49	8	3
1955-9	1	—	45-54	16	8	50-59	4	—
			55-64	5	1	60-69	1	—
						70-79	1	—
						80-89	8	2
						N.K.		
Total	109	32	Total	109	32	Total	109	32

* 10 patients with permanent foreign residence (current status unknown) and 22 patients who died within one year after Thorotrast administration.

under investigation. These also have been excluded (see Table I), and the present report on the long-term effects of the retention of thorium relates to the remaining 109 patients. Information on the time of injection, the age-distribution at injection, and the volume of Thorotrast administered is given in Table I for both this long-term study group and the 32 "exclusions" already noted.

Mortality

The present study has established the subsequent death of 45 (41%) of the 109 survivors at one year. Table II gives details of the status of these 109 individuals in relation to the diagnosis at the time of investigation, the deaths being divided into those from cerebrovascular disease, those from cancer, and those from other causes. Twenty-seven deaths were due to cerebrovascular disease, all of them among the 98 patients coming under this same classification at the time Thorotrast was administered. The other notable cause of death was

TABLE II.—Initial Diagnosis and Current Status (1 January 1966 or Later) of 109 Patients Receiving Thorotrast by Intra-arterial Injection

Initial Diagnosis	Current Status				Total Patients
	Deaths Due to			Alive	
	Cerebrovascular Disease	Other Disease (Non-cancer)	Cancer		
Aneurysm	8	2	6	26	42
Subarachnoid haemorrhage	8	1	1	21	31
Angiomata, etc.	7	1	3	7	18
Other cerebrovascular disease	4	—	—	3	7
All cerebrovascular disease	27	4	10	57	98
Other diagnoses	—	—	4	7	11
Total	27	4	14	64	109

cancer, with 14 deaths, including 12 among patients who died from malignant disease other than that for which Thorotrast was administered.² These 12 patients do not differ in terms of the volume of Thorotrast injected from those patients who died from diseases other than cancer or from those who survived (Table III). The salient features in their clinical histories are summarized in Table IV.

Apart from one female patient (Case 9) dying from breast cancer at an interval of four years the latent period from the injection of Thorotrast and subsequent death from malignant disease varied between 14 and 31 years (mean 21 years). A comparison between observed and "expected" deaths from cancer and from causes other than cancer is made in Table V, the expected deaths being calculated by applying age- and

² Two patients died from cerebral tumours four years and six years respectively after the use of Thorotrast for investigation of the tumour.

TABLE IV.—Details of 12 Thorotrast (Intra-arterial) Patients who Died from Malignant Disease

Case No.	Sex	Thorotrast Injection				Age at Death (Years)	Interval between Injection and Death (Years)	Cause of Death	Necropsy
		Year	Age (Years)	Lesion Under Investigation	Amount Injected (ml.)				
1*†	F	1942	50	Cerebral aneurysm	15	65	15	Thrombocytopenic purpura (onset 1 year before death). Giant follicular lymphoma	Yes
2*	M	1933	24	Meningioma	24	50	26	Primary carcinoma intrahepatic bile ducts	Yes
3	M	1947	41	Cerebral aneurysm	55	55	14	Carcinomatosis ? primary in bronchus	No
4*†	F	1933	23	Cerebral aneurysm	25	47	24	Primary carcinoma intrahepatic bile ducts	Yes
5	M	1944	32	Subarachnoid haemorrhage	18	53	21	Carcinoma bronchus (onset 7 years before death). Carcinomatosis	No
6*	M	1936	21	Cerebral angioma	75	44	23	Primary carcinoma intrahepatic bile ducts	Yes
7	M	1934	49	Cerebral aneurysm	18	80	31	Pancytopenia. Carcinoma of bronchus	Yes
8	M	1946	41	Osteochondroma sphenoidal sinus	20	61	20	Seminoma testis (onset 8 years before death). Oat-cell carcinoma bronchus (1 year before death). (? Haemangioendothelioma)	No
9	F	1943	45	Cerebral aneurysm	26	49	4	Adenocarcinoma breast (onset 2 years before death)	No
10	F	1944	22	Cerebral angioma	34	41	19	Haemangioendothelioma liver and spleen	Yes
11	M	1944	38	Cerebral aneurysm	38	52	14	Mucin secreting adenocarcinoma of pancreas	Yes
12	F	1942	38	Cerebral angioma	16	56	18	Anaplastic carcinoma breast	No

* Previously recorded by Ascroft and Maccabe (1962). † Reported in detail by Gardner and Ogilvie (1959).

sex-specific national (Scotland) mortality rates to the person-years experienced by Thorotrast patients tabulated by age (five-year groups) and sex for each of the seven quinquennial periods between 1931 and 1965.

TABLE III.—Distribution of 109 Thorotrast (Intra-arterial) Patients by Volume Injected and Current Status (1 January 1966 or Later)

Volume of Thorotrast Injection (ml.)	Current Status						All Patients	
	Cancer Deaths		Other Deaths		Alive			
	No.	%	No.	%	No.	%	No.	%
10-29 ..	8	12	22*	32	38	56	68	100
30+ ..	4	12	9	27	20	61	33	100
Not known	—		2*		6		8	
Total	12	11	33	30	64	59	109	100

* Includes one death due to progressive cerebral tumour which had been investigated by Thorotrast.

TABLE V.—Comparison Between Observed* and Expected Mortality (to 31 December 1965) Among Thorotrast Study Group

Person-Years Experience	Deaths			
	Observed	Non-cancer	Cancer	Total
2,032.5	Expected Ratio O/E	30.0 12.1 2.5	13.0 3.6 3.6	43.0* 15.7 2.7

* Two deaths (one due to cancer) occurring in 1966 have been excluded from this comparison.

Morbidity

Significant deposition of thorium in the soft tissues of the neck is known to have occurred in eight patients, in two of whom it was bilateral (Table VI). Five of these patients had

TABLE VI.—Details of Eight Patients with Perivascular Deposition of Thorotrast

Case No.	Sex	Thorotrast Administered	Onset	Thorotrastoma
				Signs and Symptoms
4	F	1933	—	Palpable mass on right side of neck. Patient died in 1957 (see Table IV)
13	F	1934	1965	Left recurrent laryngeal nerve palsy. Dysphagia due to spontaneous haematoma in relation to thorotrastoma
14	F	1943	1947	Dysphagia due to oesophageal stricture. Raynaud's phenomenon.
15	F	1943	1966	Left recurrent laryngeal nerve palsy
9	F	1943	1962	Right recurrent laryngeal nerve palsy
			1944	Large mass discovered on routine x-ray examination—no symptoms. Patient died in 1947 (see Table IV)
16	F	1947	1954	Bilateral deposits. Paraesthesiae of ulnar nerve distribution. Symptoms of left then right-sided axillary artery insufficiency—i.e., cold blue hand, disappearance of radial pulse*
17	F	1947	—	Bilateral deposits. Fibrosis right side of neck. No symptoms
18	M	1947	1962	Right recurrent laryngeal nerve palsy. Stricture of oesophagus on barium swallow

* This patient died in 1959. At necropsy both subclavian and axillary arteries were patent.

symptoms, including four who suffered recurrent laryngeal-nerve palsies (Cases 13, 14, 15, and 18). The most severely affected patient (Case 14) developed an oesophageal stricture four years after arteriography as a result of thorium tracking into the superior mediastinum (Fig. 1). Routine x-ray films of the neck showed two further instances of small perivascular deposits which were not causing symptoms 26 and 30 years respectively after injection, and it is also known that an earlier x-ray survey among patients in the present series detected some thorium in the neck of 18 (30%) out of 60 cases examined.

Details of changes in the peripheral blood which are suggestive of splenic atrophy (Langlands and Williamson, 1967), of an apparent increase in the incidence of thyroid disease (Langlands and Hermann, 1967), and of radiation-induced chromosome damage (Buckton *et al.*, 1967) in this series of patients have been published elsewhere.

Results of Thorotrast Injected into the Ventricular System or Subarachnoid Space

Between 1935 and 1941 27 patients are known to have received Thorotrast directly into the cerebrospinal fluid spaces

FIG. 1.—Barium swallow (1966) showing an oesophageal stricture, the result of thorium which has tracked into the superior mediastinum (Case 14).

in the course of ventriculography or lumbar myelography. Seventeen of the patients died within five years, all as a direct result of the disease bringing them under investigation and without any evidence that the course of events had been affected by the presence of thorium in the central nervous system. The present report relates to the remaining 10 patients, nine of whom have since died at intervals ranging from 7 to 32 years after Thorotrast administration (Table VII).

In contrast with the pattern after intra-arterial injection of Thorotrast, the major sequel of injection direct into the cerebrospinal fluid pathways has been the development of a progressive lesion of the cauda equina in 7 out of 10 patients at an interval varying between 6 and 16 years (mean 12 years). Disturbance of micturition progressing to complete incontinence was common to all, and muscular weakness (5 patients) and pain or other abnormal sensation (four patients) were other main features of the syndrome. Six of the seven patients affected are now dead. Three (Cases 22, 23, and 24) died from progressive renal failure and infection secondary to the neurological lesion, one (Case 21) from a pulmonary embolus after uretero-ileostomy, and one (Case 19) from uraemia. The other death was due to massive pulmonary embolus (Case 20).

Available x-ray films clearly defined the association between thorium retention and the development of a cauda equina lesion. In all seven patients the proximal level of neurological deficits appeared to correspond closely with the outline of thorium in the sacral cul-de-sac (Fig 2). Findings at operation or necropsy, available for four cases, confirmed and extended those from x-ray film and showed a gross thickening of the dura and arachnoid with widespread deposition of thorium particles. The distal subarachnoid space was obliterated, marked fibrosis was present both around and within the involved nerve roots, and actual necrosis of the dura and nerve roots was sometimes seen.

Positive evidence of cauda equina involvement was lacking for only three of the 10 patients surviving more than five years. In one of these cases (Case 26, Fig. 3) it is certain that no thorium reached the sacral cul-de-sac. At the time of ventriculography the patient had a completely blocked fourth ventricle and subsequently required a re-exploration to free adhesions. She remained free, until death 32 years later, of any symptoms referable to the cauda equina. The incomplete clinical and radiological information available for the remaining two patients (Cases 27 and 28), while essentially negative, is inadequate for any firm exclusion of the possibility of spinal complications.

TABLE VII.—*Details of 10 Patients who Survived for 5 Years or More After Injection of Thorotrast into the Ventricular System or Subarachnoid Space*

Case No.	Sex	Thorotrast Injection				Total Follow-up (Years)	Complications Due to Thorium		Notes
		Year	Age (Years)	Lesion Under Investigation	Amount (ml.)		Interval to Onset (Years)	Signs and Symptoms	
19	M	1935	26	Traumatic epilepsy	6	22	6	Incontinence of urine, sensory and motor impairment all sacral roots	Died 1957. Uraemia and cardiac failure
20	F	1935	45	Meningioma	7	24	15	Incontinence of urine, bilateral sacral and sensory and motor involvement	Died 1959. Massive pulmonary embolus
21	F	1937	37	Cranio-pharyngioma	5	19	16	Incontinence of urine, weakness of perineal muscles and anal sphincter	Died 1956. Pulmonary embolus after ureteroileostomy for incontinence
22	M	1937	31	Cerebellar haemangioblastoma (myelogram)	10	18	16	Incontinence of urine	Died 1955. Renal failure
23	M	1931	30	Lumbar disc protrusion	3	23	10	Frequency and incontinence of urine, bilateral sacral and motor involvement	Died 1964. Renal failure
24	F	1935	11	"Meningeal hydrops"	N.K.	7	6	Incontinence of urine	Death from renal failure
25	M	1937	19	Cerebral thrombophlebitis	6	30	8	Incontinence of urine, bilateral motor and sensory involvement of sacral roots. Left-sided deafness and facial palsy	Alive 1967. Clinical evidence of gradually increasing neurological deficit
26	F	1935	7	Ependymoblastoma of fourth ventricle	6	32	—	Nil	Died 1967 from bronchopneumonia after many years in mental hospital
27	M	1935	35	Meningoencephalitis	6	8	—	None known	Died 1943. Influenza and myocarditis
28	M	1937	60	Acoustic neuroma	16	20	—	None known	Frequency of micturition leading to prostatectomy 1953. Died 1957 from cerebral thrombosis

In addition to sacral cul-de-sac deposits all nine ventriculography cases had residual intracranial deposits of thorium, usually scattered in the ventricles, basal cisterns, and sulci. With the passage of time these deposits appeared to become sharper and more discrete. In spite of the presence of thorium in every case, however, only one (Case 25) provided evidence of possible local complications within the cranium. This patient developed increasing weakness of the left side of the face, with deafness, eight years after ventriculography. Skull x-ray films showed some heavy deposits of thorium in the left lateral recess of the cerebellum. The patient, who also had involvement of the cauda equina, is the only surviving member of the group.

Discussion

Since the original description by MacMahon *et al.* (1947) of the development of a tumour of the liver after Thorotrast administration numerous case reports have established in a

FIG. 2.—Lumbar spine (1946) showing retention of thorium in the sacral cul-de-sac 11 years after ventriculography with 6 ml. of Thorotrast (Case 19).

FIG. 3.—Lateral skull (1966) showing retention of large quantities of thorium, mainly in the ventricular system, in a patient who did not develop a cauda equina lesion (Case 26).

qualitative way the long-term hazards of thorium retention. Major difficulties, however, exist in attempts to obtain more quantitative assessments both of the morbidity and the risk of the subsequent development of malignant disease by the study of series of patients. Apart from the problems inherent in the calculation of tissue dosage from a non-uniformly distributed isotope, the two most important considerations in this context are the highly selected nature of the population under study and the fact that significant numbers of patients may be untraced. For example, in the largest series reported (da Silva Horta *et al.*, 1965) more than half of the 2,377 individuals known to have received Thorotrast were not traced.

The present series was defined by the use of Thorotrast in the years 1926–55 for the clinical investigation of patients in a department of surgical neurology. At that time the lesions under investigation affected mainly the cerebrovascular system—for example, congenital arterial aneurysm, subarachnoid haemorrhage—and this predominance is reflected both in the high initial mortality (22 deaths within one year of Thorotrast administration) and in the subsequent mortality experience of the 109 survivors at one year. Any quantitative assessment of mortality in the series is necessarily handicapped by these considerations, and the major biases involved in comparison (Table V) between observed and “expected” are recognized. It is, for example, scarcely surprising that on this assessment mortality among patients in this series appears to be almost three times greater than expected at standard rates, and that this excess is equally evident for deaths from all causes (observed 43, expected 15.7) and for deaths from diseases other than malignant disease (observed 30, expected 12.1). On the other hand, the admittedly heavy weighting of the series with patients with pre-existing disease of the cerebrovascular system does not readily explain the group's subsequent experience of malignant disease. It seems likely that the excess mortality from this cause (observed 13, expected 3.6) more directly reflects the risk from Thorotrast *per se*, a risk which is perhaps minimized by the excess of deaths from cerebrovascular disease at a mean interval from Thorotrast administration, which is seven years earlier than that for cases developing cancer.

Though the numbers in the present series are insufficient to permit any detailed analysis of patients dying from cancer, in terms of primary site it is noteworthy that four primary liver tumours occurred in the small population under study. While there were also three (possibly four) lung tumours, lung is a common site of cancer in the general population. Three of the liver tumours (Cases 2, 4, and 6) were primary carcinomas of the intrahepatic bile ducts and one (Case 10) was a haemangioendothelioma. In a further case (No. 8) the gross appearances of haemorrhagic liver nodules at operation shortly before death (certified as due to an oat-cell carcinoma of the bronchus) also suggested haemangioendotheliomatous change, but no biopsy was taken at operation and necropsy was not carried out. The four patients with confirmed liver tumours had similar characteristics both with respect to age at injection (24, 23, 21, and 22 years respectively) and the interval between injection and subsequent death (26, 24, 23, and 19 years respectively). There is, however, a wider variation in the volume of Thorotrast received (24, 25, 75, and 34 ml. respectively) at the time of initial investigation, and, while our data as a whole provide no convincing evidence of any direct relation between the volume of Thorotrast injected and the subsequent fate of the patient (Table III), the limited data on liver cancer are at least consistent with such a relation. Thus 2 out of 68 patients receiving less than 30 ml. of Thorotrast have developed a liver tumour (1 in 34) compared with 1 in 20 receiving 30 to 39 ml. and 1 out of 13 receiving at least 40 ml. of Thorotrast.

The progressive fibrosis which follows the perivascular deposition of thorium or its direct injection into the cerebro-

spinal fluid is an important cause of morbidity. The 10 significant perivascular deposits (eight patients) noted in the present series represent a minimum assessment of the occurrence of the complication, since information on this aspect was available in only a proportion of cases. Of considerably greater significance in this context was the development of progressive cauda equina lesions after direct injection of Thorotrast into cerebrospinal fluid spaces. After injection into the ventricles thorium diffuses rapidly and spreads throughout the cerebrospinal system. It tends to collect in the more dependent parts of the ventricles, in the basal cisterns, on the choroid plexus, and most of all in the sacral cul-de-sac. The particles of thorium settle out and gradually become incorporated within the ependymal lining of the ventricles and the surface layer of the arachnoid membrane.

The serious sequelae after deposition in the sacral cul-de-sac have been amply demonstrated in the present series. Seven out of 10 patients surviving five years after Thorotrast administration developed progressive cauda equina lesions. Four (possibly five) of six subsequent deaths among these patients were the direct consequence of interference with bladder innervation. Taken as a whole the evidence pointing to thorium as the cause of cauda equina lesions appears conclusive and is consistent with the previous demonstration (Reeves and Stuck, 1938) of other hazards, such as hydrocephalus and damage to cranial and spinal nerve roots, after the prolonged exposure of neural tissue to thorium, and with similar cases which have been described by Hughes (1953) and by Dale and Love (1967). It is quite plain that Thorotrast should never be injected into the cerebrospinal fluid or into a cavity likely to communicate with cerebrospinal pathways. It is also fortunate that ventriculography or myelography with Thorotrast, never used with confidence, was so soon and so firmly abandoned.

Summary

All patients (168) known to have received Thorotrast in the course of clinical investigation in a department of surgical

neurology have been followed up and their current or final status at 1 January 1966 or later established. A total of 137 patients received Thorotrast by intra-arterial injection during the years 1933 to 1955. While their subsequent mortality experience reflected the diseases, mainly cerebrovascular, bringing them under investigation, it was notable that there were 12 deaths from cancer (including four primary liver tumours) among the 109 who survived more than one year after Thorotrast administration.

Between 1935 and 1941 27 patients received Thorotrast by direct injection into the ventricular system or subarachnoid space. Seventeen of these patients died within five years as a direct result of the disease bringing them under investigation. Seven of the 10 five-year survivors subsequently developed progressive cauda equina lesions, six of which have proved fatal.

We are pleased to acknowledge our indebtedness to Professor Norman Dott for generous co-operation and help throughout and to Dr. Kate Hermann for her continued interest in the investigation. We are deeply indebted also to the late Mr. Peter Ascroft for his careful definition of the population under study. Miss A. Fotheringham, Mrs. E. Goldenberg, and Miss C. M. Jones undertook the considerable task of tracing patients. Our thanks are due to them and the many doctors who provided information about their own patients.

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Acquired Cytomegalovirus Infection*

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The role of the cytomegalovirus group as pathogen for the foetus has been well documented since the isolation of the first cytomegalovirus strains and the consequent development of virological methods of diagnosing the disease. Infection in utero is contracted from a mother, generally without clinically recognized disease. The congenital infection has been described as a generalized disease which may resemble congenital toxoplasmosis, neonatal hepatitis, or neonatal sepsis. It has usually been regarded as a very serious illness, but recent observations indicate that various degrees of severity may occur. Jaundice, petechial rash, hepatosplenomegaly, microcephaly, and mental retardation are common manifestations (Weller and Hanshaw, 1962).

Little is known about the clinical features of postnatally contracted cytomegalovirus infections. Before the use of virological techniques the recognition of these infections depended on the microscopical detection of typical inclusion-bearing cells. Such cells were found at post-mortem examination of adults with conditions complicating malignant and chronic

debilitating diseases. Wong and Warner (1962) reported 14 such cases and reviewed 41 others in the literature. Diseases known to alter the resistance of the host to infection were prevalent and the majority of cases were treated with corticosteroids. The possibility of a reactivation of dormant virus has been discussed. Common clinical manifestations of such secondary cytomegalovirus infections were ulcerative gastroenteritis, pneumonitis, or generalized disseminated disease. The complication of cytomegalovirus infection in debilitating diseases and in immunodepressive therapy has now in some cases been verified by means of virological techniques (Hanshaw and Weller, 1961; Carlström, 1966; Craighead *et al.*, 1967).

Reports on postnatally contracted cytomegalovirus infection occurring without connexion with debilitating diseases are scarce, but the development of virological diagnostic methods

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